Anesthetic management of the cocaine abuse patient
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Cocaine is an extremely addictive local anesthetic which can produce stimulation of the sympathetic nervous system due to the inhibition of catecholamine reuptake at the synaptic junction. Because of the rapid metabolism of cocaine, the probability of a patient presenting to the operating room with acute intoxication is unlikely. However, the physiological effects of chronic cocaine abuse on various organ systems have an impact on anesthesia management.

A preoperative review of major organ systems is essential. Selective beta1 antagonists (i.e., esmolol) may need to be titrated with a direct vasodilator (i.e., nitroprusside) to manage hypertension and tachycardia. The nonselective beta antagonist effects of labetalol are much more potent than its alpha antagonist effects, which could result in unopposed alpha vasoconstriction. In addition, the equal affinity of the alpha adrenergic antagonist, phentolamine, for both alpha1 and alpha2 receptors may result in significant tachycardia. Nitroglycerin has also been used in management of hypertension associated with coronary vasoconstriction. There is controversy regarding management of ventricular dysrhythmias and asystole. Lidocaine is an amide local anesthetic that may have addictive effects, in the presence of cocaine, which may lower the seizure threshold. In addition, the use of epinephrine to treat asystole is controversial in the presence of a state of excess catecholamines induced by cocaine.

General anesthesia may include barbiturates, nitrous oxide, and opioids. Inhalational agents may be used with caution due to their myocardial depressant effects. Regional anesthesia may be a good choice if coagulopathies and hypovolemia are corrected before the procedure.

Key words: Cocaine, esmolol, labetolol, propranolol, substance abuse.

Cocaine is an alkaloid extract from the leaves of the Erythroxylon coca bush. For centuries, the natives of Bolivia and Peru have chewed the leaves for their stimulant effect. In 1868, cocaine was recognized for its local anesthetic effects. By the 1880s, physicians became aware of the addictive properties of cocaine through self-experimentation. Despite laws prohibiting the use of cocaine, it has been estimated that 30 million Americans have used cocaine, five million use it regularly, and another 5,000 use it each day for their first time. Myths about cocaine being nonaddictive and physically benign have contributed to the increased use of cocaine, making it the most popular illegal drug in the United States. In truth, cocaine produces
euphoria that is followed by depression and craving for more cocaine, resulting in psychological addiction.

Chewing coca leaves is not as toxic as using the processed form, cocaine hydrochloride. Cocaine hydrochloride is absorbed through any mucous membrane but the most popular route is intranasal insufflation, or “snorting.” Cocaine is smoked in the “freebase” and “crack” forms. Relatively pure cocaine crystals are prepared by actual users of “freebase.” “Freebase” users are at risk for burns when some of the highly flammable ether is extracted in addition to the cocaine crystals.1 “Crack,” or “rock,” is usually sold premade and ready to smoke. It has become more popular since it is less expensive, more available, eliminates the risk of flammability, and is simpler to prepare. About 30% of cocaine users have smoked it and 10% have injected it.1 When injected, cocaine may be mixed with heroin and is called a “speedball.”2

Cerebral effects, called a “rush” or “flash,” occur within 6 to 8 seconds after smoking cocaine and last 20 minutes. Intravenous (IV) cocaine reaches the brain in 15 seconds. Smoking cocaine produces peak blood levels that are 60% of the same dose given IV. Nasal insufflation produces a “high” in 5 minutes and is not as intense as the “rush” produced by smoking or injecting. “Snorting” peaks in 30-60 minutes and lasts 90 minutes. Blood levels achieved are about 20% to 60% of the same dose given IV.1

Regardless of route, plasma cholinesterase metabolizes more than 80% of the cocaine into two inactive metabolites, ecgonine methyl ester and benzoylecgonine.1 Liver cholinesterase metabolizes 10% of the cocaine to norcocaine, an active metabolite that produces actions similar to cocaine, may account for prolonged effects and may increase the risk of cocaine-induced complications. Abnormal plasma cholinesterase may lead to greater metabolism of cocaine by liver cholinesterase into norcocaine, resulting in higher and prolonged levels of cocaine.3 The elimination half-life of cocaine is approximately 30-60 minutes after IV use and 60-90 minutes after nasal insufflation.4

Preoperative considerations

Drug history. Since polysubstance abuse is common, a drug history should include specific questioning about the timing, amount, frequency, and route of all drugs taken in the last 24 hours, as well as length of addiction.2 Informing the abuser of potential drug interactions with anesthetics may prompt honest reporting of drug use.

Drug testing, although not required, may help identify what drugs are present. The usefulness of blood testing is limited because of the rapid metabolism of cocaine. Urine testing for cocaine metabolites is much more useful with 99.2% accuracy, 96% sensitivity, and 100% specificity.1 Urine testing can detect cocaine metabolites up to 6 days after a single dose and up to 10 or 20 days after high dose, long-term use.1

Cardiovascular effects. Cocaine blocks the reuptake of norepinephrine, dopamine, and serotonin at the synaptic junctions producing an excess of transmitter at the postsynaptic receptor sites. The effects that occur are due to sympathetic stimulation. Cardiovascular effects of acute cocaine use manifest as hypertension, tachycardia, dysrhythmias, myocardial infarction (MI), cardiomyopathy, or aortic rupture.3 Cocaine-induced MI may manifest within minutes after cocaine use or up to 18 hours later.1 Patients who have had a cocaine-induced MI tend to be younger, an average age of 31 years, than patients with coronary artery disease. About one third of these cocaine-using patients have been found to have normal coronary arteries on angiography. The other two thirds had atherosclerotic disease, despite their young age. It is hypothesized that cocaine alters the endothelium of coronary arteries, promotes atherogenesis, impairs vasodilation, and acts as a myocardial depressant. Similar to other local anesthetics, cocaine produces negative inotropic effects.2 Silent ischemia may be evidenced by electrocardiogram (ECG) changes in the first week of withdrawal. Thus, a 12-lead ECG is essential for the cocaine abuser. Transesophageal echocardiography may help evaluate cardiac function, aortic dissection, or valvular abscess.

Respiratory effects associated with smoking cocaine may include bronchospasm, pulmonary edema, pulmonary hemorrhage, and “crack lung.” Crack lung is a cocaine-induced lung disease characterized by diffuse alveolar infiltrates on chest x-ray, eosinophilia, and alveolar hemorrhage that may manifest with chest pain, hemoptysis, and dyspnea.8 Pneumothorax, pneumomediastinum, and pneumopericardium may occur after freebase smoking that requires forced, prolonged inspiration with the Valsalva’s maneuver in order to increase cocaine distribution in the pulmonary circulation and augment the intensity of the drug’s effect. Some users go so far as to have another person forcefully blow smoke into their mouths. This mouth-to-mouth positive pressure by another person may cause further barotrauma.1 Chest x-rays may help to rule out pneumothorax as the etiology of chest pain or dyspnea and to identify infiltrates, scarring, or pulmonary edema. Pulmo-
nary function tests may help to differentiate between obstructive or restrictive lung disease.

- **Neurologic effects** are associated with higher cocaine levels achieved by smoking or injecting cocaine and may present as headaches, hyperthermia, seizures, subarachnoid hemorrhage, intracranial hemorrhage, cerebral infarction, cerebral vasospasm, or cerebral atrophy. A cocaine-induced headache associated with a decrease in level of consciousness, nausea, vomiting, or neck stiffness could be symptoms of intracranial hemorrhage. A computed tomography scan is warranted.

- **Hematologic effect.** The major hematologic effect seen with cocaine abuse is thrombocytopenia that may be associated with induction of platelet antibodies or bone marrow suppression. Thrombocytopenia does not appear to be related to the route of administration and, in severe situations, may produce prolonged bleeding.

- **Renal complication.** The most common renal complication of cocaine abuse is rhabdomyolysis, a syndrome resulting from skeletal muscle injury and the release of myoglobin into the bloodstream. Rhabdomyolysis may be due to vasoconstriction, ischemia, hyperthermia, or a direct toxic effect of cocaine on the muscle cells. The classic signs and symptoms include nausea, vomiting, myalgias, and muscle swelling. Elevated serum creatine kinase levels and myoglobinuria may be present. Elevated uric acid levels, hyperkalemia, hypocalcemia, and metabolic acidosis indicate more advanced rhabdomyolysis associated with renal failure or hepatic damage.

- **Gastrointestinal disorders.** Cocaine-induced gastrointestinal disorders include weight loss, colitis, gastroduodenal perforations, or intestinal ischemia. Intestinal ischemia should always be suspected when a cocaine abuser has severe abdominal pain and an elevated white blood cell count. An abdominal x-ray is mandatory in suspected "body packers," persons who ingest packets of cocaine in order to conceal them. If the wrapping deteriorates while in the body, acute toxicity can occur.

- **Head and neck.** Changes of the head and neck area may present as chronic rhinitis, sinusitis, or ulceration of the nasal septum. Anesthesia practitioners who identify these findings during a preoperative assessment should consider the potential for cocaine abuse. Smoking cocaine can result in laryngeal burns manifested as hoarseness, stridor, or dysphagia.

- **Pregnancy.** Cocaine abuse during pregnancy is associated with an increase in morbidity and mortality of the parturient, the fetus, and the neonate. Complications of pregnancy include abruptio placenta, premature labor, precipitate delivery, and spontaneous abortion. Effects on the fetus and neonate include low birth weight, intracerebral growth retardation, neurobehavioral deficits, microcephaly, congenital abnormalities, or apnea. The major predictor of cocaine abuse in the parturient is the absence of prenatal care.

- **Infections.** Risk of infections is increased, relative to the environment in which cocaine is used. Crack cocaine use has been associated with an increased incidence of sexual risk behaviors that result in syphilis, gonorrhea, or sexually transmitted human immunodeficiency virus (HIV) infection. Sharing of needles increases the risk of IV transmission of HIV, hepatitis, or bacterial sepsis. In addition, respiratory infections or tuberculosis may be transmitted in crowded, poorly ventilated rooms.

- **Acute cocaine intoxication.** The incidence of patients presenting with acute cocaine intoxication secondary to social abuse of the drug is reduced by its rapid metabolism to primarily inactive metabolites and its short biological half-life of 1 hour. Most trauma surgeries are initiated within about 3 hours after the trauma event and possibly the last cocaine dose. Even in the hospital, some cocaine users are very adept at obtaining illicit drugs. Signs of acute cocaine intoxication include impaired judgment, hypervigilance, agitation, paranoia, syncope, mydriasis, vomiting, chest pain, tachycardia, hypertension, hyperthermia, respiratory paralysis, seizures, and death. In the absence of acute intoxication, anesthesia considerations for the chronic abuser are primarily related to the physiological changes that occur in various organ systems.

### Intraoperative anesthetic considerations

Following a thorough preoperative assessment, attempts should be made to stabilize the altered hemodynamics prior to induction. The premedication for surgery should provide enough sedation and anxiolysis while taking into account the patient's potential for increased tolerance to sedatives and narcotics.

Cocaine-induced hypertension is due to vasoconstrictions from alpha stimulation. Reduced intravascular volume may be masked by sympathetic vasoconstriction. Assessment of intravascular volume or blood loss should not be based solely on a normal blood pressure.

Controversy exists regarding the management of cocaine-induced hypertension and tachycardia. Various pharmacologic interventions have been used and include the following:

1. Propranolol, a nonselective beta adrenergic...
blocker, was advocated at one time for use to treat tachycardia because of its beta₁ blockade. However, propranolol was found to (1) worsen the myocardial depressant effect of cocaine on the left ventricle and (2) allow unopposed alpha-adrenergic stimulation manifested as increased peripheral and coronary vascular resistance due to blockade of beta₂ receptors. 20

2. Labetalol, which has selective alpha₁ and nonselective beta₁ and beta₂ antagonist properties, has been used successfully to manage hypertension. However, it has been suggested that the use of labetalol could result in an unopposed alpha effect since the nonselective beta-blocking effects are as much as seven times more potent than the alpha-blocking effects. 21

3. Phentolamine, an alpha antagonist, has been used to treat hypertension. Phentolamine inhibits vasoconstriction in response to endogenous catecholamines via the blockage of alpha₁ receptors. However, it has an equal affinity for blockage of alpha₂ receptors. This results in significant tachycardia caused by release of norepinephrine with its corresponding stimulation of beta₁ receptors. 22

4. Nitroprusside has also been documented for use in hypertension related to cocaine abuse. Vasodilation occurs in response to the release of nitric oxide as the drug decomposes. This vasodilation is associated with a reduction in arterial pressure, a modest baroreceptor-mediated increase in heart rate, and a decrease in myocardial oxygen consumption. Nitroprusside is superior to other hypotensive drugs due to its rapid onset of action, easily titratable dose to the desired effect, and lack of tachyphylaxis seen with other drugs like phentolamine. 23

5. Esmolol has been used successfully since it selectively blocks only beta₁ receptors. Esmolol appears to result in less incidence of hypertension from unopposed alpha-agonist activity than does the nonselective beta antagonist, propranolol. In addition, the short duration of action of esmolol allows for easier titration. 21 Titration of esmolol and nitroprusside infusions appears to be the optimum choice for management of hypertension and tachycardia related to the hyperadrenergic state of cocaine abuse. 24

In the past, calcium channel blockers were believed to reverse the coronary artery vasoconstriction caused by cocaine. However, researchers found that calcium channel blockade can prevent catecholamine-mediated overload of calcium into the myocytes if given before cocaine but is ineffective when the myocardial cells are already overloaded with calcium. 24 Nitroglycerin has been recommended to treat coronary vasoconstriction in patients with chest pain associated with cocaine use. Both calcium channel blockers and nitrates have also been used to manage hypertension. 25

Other ester local anesthetics, such as tetra-caine, procaine, and chlorprocaine, may augment the proconvulsive effects of cocaine. 25 Diazepam and thiopental are recommended for management and prevention of cocaine-induced seizures. 26 It has also been recommended that lidocaine, an amide local anesthetic, be avoided in treatment of ventricular arrhythmias since it may further reduce the seizure threshold in the presence of cocaine. 27 Brevetilum should be used with caution in treating ventricular arrhythmias associated with cocaine abuse since it causes an initial, but transient, release of norepinephrine which can potentiate hypertension and tachycardia. 28 Propranolol has been recommended by some for treatment of ventricular arrhythmias in the absence of hypertension. 19 The use of epinephrine to treat asystole secondary to cocaine use is controversial since both drugs produce a hyperadrenergic state. 29 However, the literature does not suggest an alternative to epinephrine in the event of asystole.

The use of volatile anesthetics for general anesthesia may sensitize the myocardium to the dysrhythmic effects of catecholamines. 30 In addition, cocaine may cause an increased minimum alveolar concentration (MAC) requirement for volatile agents. 23 The use of halothane, a strong myocardial depressant, is not recommended since it sensitizes the myocardium to the effects of catecholamine, thereby inducing various types of arrhythmias. 4 12 Although enflurane is less of a myocardial depressant, it may produce seizures at high concentrations of greater than 2 MAC, especially in the presence of hypocapnia. 28 Theoretically, isoflurane is the best choice for a volatile agent since it has less myocardial depressant effects and has a high threshold to induce cardiac arrhythmias. 12 Some authors argue that deep levels of general anesthesia inhibit the adrenal release of catecholamine, thereby reducing the dysrhythmic potential. 20 It is suggested that deep general anesthesia, using nitrous oxide and any of these volatile agents, may be used in the presence of cocaine abuse. 30 The advantages of increasing seizure threshold with thiopental and decreasing the heart rate with fentanyl make these agents useful in general anesthesia for cocaine patients. 30 Ketamine should be avoided because of its sympathomimetic effects. 25 Neuromuscular blocking agents, such as succinylcholine, vecuronium, doxacurium, and d-Tubocurarine may be used. Gallamine and pancuronium should be used with caution in the pres-
ence of tachycardia due to their sympathomimetic effects.30

Regional anesthesia has been used successfully in the management of parturients who have a history of cocaine abuse.3 Prior to performing the procedure, consideration should be given to the potential for coagulopathies, sepsis, HIV infections, and hypovolemia.31 A platelet count should be evaluated before the procedure to rule out thrombocytopenia related to cocaine abuse.10 Uncorrected coagulation defects are an absolute contraindication to epidural or spinal anesthesia.32 The potential for bacteremia, sepsis, or HIV infection is also important to consider prior to regional anesthesia. Although localized infection at the puncture site is an absolute contraindication, regional anesthesia can be performed with caution in the presence of bacteremia and sepsis.32 No adverse outcomes have been reported when regional anesthesia was performed on HIV patients, suggesting that the presence of HIV infection is not an absolute contraindication to regional anesthesia.4

A study done on the risks of epidural anesthesia on parturients who used cocaine during their pregnancy suggested that epidural anesthesia be used with caution in these patients.33 This study noted a significant increase in the incidence of hypotension and need for supplemental IV narcotics.33 It has been suggested that the segmental level of epidural anesthesia should be raised gradually by titration of the local anesthetic dose and by hydration to prevent hypotension.4 The anesthesia practitioner should also consider the risks of potentiation of sympathomimetic effects of cocaine by the use of epinephrine and the reduction in the seizure threshold by the use of additional local anesthetics. At times, the benefits of regional anesthesia may outweigh the risks.32

Chronic use of cocaine may also deplete the central nervous system stores of dopamine and serotonin.34 In the event of catecholamine depletion, there may be a decreased MAC requirement for volatile agents and hypotension may be manifested. Direct acting sympathomimetics such as phylephrine or epinephrine are recommended to treat hypotension in these patients.32 The incidence of extrapyramidal side effects of dopaminergic antagonists, such as methyldopa, haloperidol, droperidol, and metoclopamide, may be increased because of the dopamine depletion that may occur with chronic cocaine use.30 These agents should be used with caution. However, haloperidol continues to be recommended as the first choice in managing psychotic symptoms.30 Diphenhydramine may be used to treat dystonic reactions that occur.

Summary

Cocaine is the most popular illegal drug used in the United States and has been associated with thousands of deaths in the last 10 years.30 In order to manage the cocaine user, the anesthesia practitioner should be knowledgeable regarding the physiological effects of chronic abuse, as well as management of acute intoxication. Severe cardiovascular effects are the major concern and may be treated by titration of esmolol and nitroprusside infusions. General anesthesia may best be managed by the use of a nitrous-narcotic technique or deep planes of anesthesia. The use of regional anesthesia is appropriate if coagulopathies and hypovolemic states are corrected. Despite the recommendations that regional anesthesia may be performed in patients with bacteremia and HIV infections, the risks to the practitioners should remind us of the necessity for universal precautions.

REFERENCES


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